

The adverse respiratory events reported here in children during general anesthesia add significant validity to accumulating evidence on the risks of ETS in children. These risks include alteration of pulmonary function, exacerbation of asthmatic attacks, level of bronchial response in asthma, wheezing in young children, upper and lower respiratory tract illness, and middle ear effusion.

Any educational effort aimed at encouraging smokers to quit and to protect their children from ETS is an uphill task. The trigger event that could be used in this circumstance would be the reporting by anesthesiologists to surgeons and pediatricians of the patients' families of the occurrence of adverse respiratory effects during general anesthesia and the likelihood that they were associated with exposure to ETS. I have long advocated that on the initial assessment of new pediatric patients, history-taking include exposure to environmental tobacco smoke. Parents, especially new first-time parents, want to do the right thing for their children, and this is the best time for a physician's

admonition about ETS exposure to make an impact. There are many simple preventive measures that never occur to parents that can save lives, such as suggesting to a parent who believes a handgun must be kept in the home that the bullets for the weapon should be hidden in another place.

When all of the detrimental effects of ETS on children are considered, especially those in asthmatic children, smoking in households where there are children may well become the next issue in child abuse.

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## Reference

1. Skolnick ET, Vomvolakis MA, Buck KA, Mannino SF, Sun LS: Exposure to environmental tobacco smoke and the risk of adverse respiratory events in children receiving general anesthesia. *ANESTHESIOLOGY* 1998;88:1144-53

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1998; 88:1142-3  
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## How Does $\mu$ -Opioid Receptor Blockade Work in Addicted Patients?

WHEN I first heard the CNN broadcast and then later read the *New York Times* story about patients who receive general anesthesia to break opioid addiction, my immediate response was, "Well, sure it will work—until the patient wakes up. An addict cannot take drugs while asleep, but the basic biochemistry of his or her brain will not change." But on further consideration, I think it is possible that the mechanism that produces the unconscious state during general anesthesia and prevents an addict from "feeling" may

indeed permanently alter brain biochemistry. A crazy thought? In this issue of *ANESTHESIOLOGY*, Kienbaum *et al.*<sup>1</sup> describe the rapid detoxification of addicts during general anesthesia with the use of  $\mu$ -opioid receptor blockade. The investigators do not speculate about the mechanism behind this therapy. My question is, does the therapy really work? To call the therapy effective, Kienbaum *et al.* must show that in the long term, brain biochemistry in their subjects was changed. Or failing that, proof is needed that the subjects abstained from drugs long term.

The study, which presents one therapy to reverse the biochemistry of addiction, may teach us something about the biochemistry of anesthesia. We know that opioids and general anesthetics suppress the autonomic nervous system. Many studies have suggested that these drugs work synergistically rather than additively. In the 1970s, I spent a year at NIH trying to confirm the changes in brain receptors caused by opi-

This Editorial View accompanies the following article: Kienbaum P, Thurauf N, Michel MC, Scherbaum N, Gastpar M, Peters J: Profound increase in epinephrine plasma concentration and cardiovascular stimulation following  $\mu$ -opioid receptor blockade in opioid-addicted patients during barbiturate anesthesia for acute detoxification. *ANESTHESIOLOGY* 1998; 88:1154-61.



oids. I was searching for changes that would make a receptor more "appealing" to volatile anesthetic effects or *vice versa*. I was never able to prove my hypothesis during that year.

The article by Kienbaum *et al.* once again reminds us of the strong interaction between opioid agents and anesthetics. The synergy may exist on a molecular biologic level, one strong clue to the mechanism of general anesthesia, or at least to the cardiovascular effects and control mechanisms that operate during general anesthesia. Thus this article, like many in the anesthesia literature, leads us more toward reexamination of questions than evaluation of answers.

O'Connor and Kosten<sup>2</sup> recently reviewed the scientific literature on rapid detoxification techniques in addicts. They indicated the limitations of the available studies: lack of randomized design, of comparison with other treatment methods, and of control groups; short follow-up periods (days, in some studies); and variation in protocols. Although we cannot endorse the therapy

for addiction described by Kienbaum *et al.* without the results of further research, we can seek to understand what this treatment tells us about opioid addiction and the mechanism of general anesthesia, two — until now — unrelated subjects.

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## References

1. Kienbaum P, Thurauf N, Michel MC, Scherbaum N, Gastpar M, Peters J: Profound increase in epinephrine plasma concentration and cardiovascular stimulation following  $\mu$ -opioid receptor blockade in opioid-addicted patients during barbiturate anesthesia for acute detoxification. *ANESTHESIOLOGY* 1998; 88:1154-61
2. O'Connor PG, Kosten TR: Rapid and ultrarapid opioid detoxification techniques. *JAMA* 1998; 279:229-34